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VASO-MOTOR NERVES OF THE HEART.

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There has come about in physiology within the last ten years the use of two new words, *anabolic* and *katabolic*. The justification for the employment of these words is mainly to be found in recent experiments made on the heart which show that certain nerves tend to help the nourishment of the heart and are in the old sense of the word, true *trophic* nerves, while certain other nerves, when active, tend to exhaust the heart and to prevent or hinder its nutrition. Experiments first made on the heart by Gaskell have since been extended to muscles and to other organs, so that the physiology of to-day is largely a question of ~~an~~ anabolic and katabolic phenomena. Investigation of the extrinsic cardiac nerves, with their ganglia, shows that although stimulation of the pneumogastric slows the pulse, or strong excitation entirely stops the heart beat, yet on the whole the nerve is a help to the heart; that if in an animal you take a feebly beating heart stimulation of the pneumogastric will, after a time, strengthen the heart. If you take the dying heart of a frog, in which the auricles are still beating, but the beat fails to pass over to the ventricle, by stimulating the pneumogastric you can often get the heart back to its normal rhythm. Often one gets the phenomenon that the auricle in a dying heart makes two beats for the ventricle's one, and finds that on stimulating the pneumogastric one gets the normal beat, one stroke of the auricle corresponding to one stroke of the ventricle; we can therefore on experimental grounds assert that the pneumogastric is essentially a trophic nerve for the heart.

The same observer (Gaskell) had also shown some few years before that, mingled with the motor fibres of an ordinary motor nerve were vaso-dilator fibres, fibres which

dilated the arteries of the muscle at the same time that the muscle fibres contracted. One can paralyze the motor fibres proper by curare, and then, on stimulating the nerve trunk, get the vascular dilatation without the muscular contraction; but normally the two things go together. Whenever a muscle contracts, be the impulse that excites the contraction voluntary or reflex, the contraction is accompanied by an impulse which acts upon the blood vessels of the muscle and for a time paralyzes their muscular coats, so that its arteries allow more blood to flow through the muscle.

It occurred to me that it would be interesting to investigate this question in connection with the nerves of the heart. We there find the vagus of which I have just spoken; and in addition the accelerator nerves which, when excited, quicken the heart's beat, and may in a sense be called the motor nerves of the heart; though, as is well known, the heart will continue to beat when all influences from outside nerves are cut off from it.

The vagus being the trophic nerve of the heart, it might be readily supposed *a priori* that it would be the nerve which contained the fibres for dilating the coronary arteries. On the other hand we have the analogy of ordinary muscle where we know that the motor nerve branches which excite the organ to activity are those which contain the vaso-dilator nerves fibres.

At the beginning of this session I asked Mr. Lingle to study this problem with me, in the physiological laboratory of the Johns Hopkins University. Our research is still far from complete, but we have, I think, reached some interesting results. Our method has been to anæsthetize the animal, open the thorax, (of course starting artificial respiration) and then by opening the pericardium to expose the heart: we then selected some little artery on the surface of the ventricle for careful observation. The best arteries are those that are in the etymological sense of the word capillaries, little arteries just as big as a hair, which one watches through a hand lens. It is quite easy to observe them, but it is not easy always to observe their changes in diameter, but by selecting a little network on the surface of the myocar-

dium and watching attentively, one can usually see any changes that occur in the diameter of its vessels.

We began by stimulating the pneumogastrics, and found that every time that the pneumogastrics were stimulated the arteries of the heart dilated; the heart became distinctly redder and the superficial arteries of the myocardium were very distinctly increased in diameter. Then the query arose which led to the most interesting point in our work.

In order to watch accurately these changing blood vessels it was necessary to stop the artificial respiration, because the lungs moving up and down, kept heaving the heart up and down: It was hard enough to watch a given vessel during the beat of the heart itself; with the respiratory movements added it was practically impossible. But the cessation of artificial respiration introduced a new factor; and we therefore tried the effects of stopping the respiration without simultaneously stimulating any nerve. Under these conditions we got *diltation* of the conorary arteries, though dyspnœa causes constriction of the arteries in every other part of the body.

The diagram I exhibit is taken from a typical experiment. The top line is the chronograph line, each notch being a second. The next is the respiration line. Toward the far end of the diagram there are parts of two respiration curves. At the level of the first vertical line the respiration stopped, the stopcock having been turned which cut the lungs off from the pump, and towards the other end of the tracing respiration commenced again, as is indicated by the ~~nerves~~. curves
The line C has one little notch in it. That notch was worked by the observer with a little electric signal and indicated when he saw a change in the blood vessels of the heart. The lowest line is a tracing of the blood pressure taken in the carotid artery, the beats being of course pulse beats.

The point I want to call attention to is this. You see that for some time after the respiration is stopped there is no rise in the blood pressure and before there is any rise in the blood pressure the observer signals that he has seen a change in the diameter of the blood vessels. That change was always

an increase. Later on comes the dyspnœic rise of blood pressure and then, artificial respiration being used again, blood pressure returns to the normal.

In our first observations as to the diameter of the cardiac arteries during commencing asphyxia, we thought their dilatation might be due to the fact that there was a general rise of blood pressure, which rise mechanically distended the coronary arteries and their branches; but, as you see, the observer always signals before there is any rise of blood pressure in the carotid; therefore the phenomenon is a true vaso-dilator one. The point of chief interest is that while in commencing suffocation the arteries of the body in general constrict, the arteries of the heart dilate. The heart arteries dilate that the last remaining oxygen shall go to that fundamental vital organ. It is one of the most beautiful preservative mechanisms that I know of in the whole physiological working of the body.